IN THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF SOUTH CAROLINA
CHARLESTON DIVISION

In Re: AQUEOUS FILM-FORMING FOAMS PRODUCTS LIABILITY LITIGATION	MDL No. 2:18-mn-2873-RMG **This Document Relates to:** *Brock Donnelly v. 3M Company et al.*, No. 2:20-cv-00209-RMG *Clinton Speers & Gail Speers v. 3M Company et al.*, No. 2:21-cv-03181-RMG *Kevin Voelker v. 3M Company et al.*, No. 2:18-cv-03438-RMG

DEFENDANTS' FIRST OMNIBUS MOTION TO EXCLUDE PLAINTIFFS' CAUSATION AND PLAUSIBILITY EXPERT TESTIMONY

TABLE OF CONTENTS

TABLE OF AUTHORITIES

Page(s)

Cases

INTRODUCTION

The Court should exclude the testimony of Plaintiffs' general causation, specific causation, and biological plausibility experts under Federal Rule of Evidence 702. For independent reasons, the reasoning or methodology underlying their testimony is not scientifically valid: (1) Dr. Joseph Braun (general causation) relied on statistically insignificant study results to reach the legally insufficient conclusion that "any amount" of exposure to PFOS and PFOA increases risk of kidney cancer; (2) Dr. Robert Bahnson (specific causation) conducted an unreliable differential etiology because he did not take serious account of Plaintiffs' recognized risk factors for kidney cancer (or failed to consider them at all) before ruling them out as an alternative cause; and (3) Dr. David Sherman (biological plausibility) ignored serum concentration disparities by at least 1,000-fold in attempting to apply data from *in vitro* and *in silico* studies to human populations. For these reasons and additional reasons discussed below, their testimony is inadmissible.

ARGUMENT

I. Dr. Braun's General Causation Opinions Should Be Excluded.

The Court should exclude the testimony of Dr. Braun, Plaintiffs' sole general causation expert, under Rule 702. "[E]pidemiologists use a two-part process for determining causation." *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs. & Prods. Liab. Litig. ("Lipitor III")*, 174 F. Supp. 3d 911, 914 (D.S.C. 2016). "First, epidemiological studies must establish an association between exposure to a [substance] and a disease." *Id.* And where plaintiffs' experts agree "there is a dose-response relationship and where there is evidence that an association no longer holds at low doses, dose certainly matters," and plaintiffs must have expert testimony that there is an association between exposure and injury at particular doses. *Id.* at 919. "Once an association has been found between exposure to an agent and development of a disease, researchers consider whether the association reflects a true cause-effect relationship." *Id.* at 916.

Dr. Braun's analysis fails at each step. *First*, Dr. Braun cannot show a relevant and statistically significant association between exposure to PFOS or PFOA and kidney cancer. *Second*, without this threshold showing of a statistically significant association, Dr. Braun cannot proceed to the next stage of evaluating actual general causation by analyzing the Bradford Hill factors. Even setting that aside, Dr. Braun's analysis is impermissibly premised on an unreliable literature review. *Finally*, Dr. Braun contravenes the central tenet of toxicology that the "dose makes the poison" by resorting to the discredited "any exposure" theory. The Court should bar Dr. Braun from testifying that exposure to PFOS or PFOA is capable of causing kidney cancer.

A. Dr. Braun's Reliance On Non-Statistically Significant Associations Between PFOS Or PFOA And Kidney Cancer Is Unreliable.

Dr. Braun's opinions should be excluded because he has not "point[ed] to a single study that shows an association" between kidney cancer and PFOS (at any exposure level) or PFOA (at relevant exposure levels) that is statistically significant, and an epidemiologist's exclusive reliance on non-statistically significant results to reach an opinion on general causation is unreliable. *Lipitor III*, 174 F. Supp. 3d at 919, 926; *see also Soldo v. Sandoz Pharms. Corp*., 244 F. Supp. 2d 434, 533 (W.D. Pa. 2003) ("[c]ourts have emphasized that epidemiologic proof must be statistically significant"; collecting cases). Like plaintiffs' general causation expert in *Lipitor III*, Dr. Braun identified only studies that either show no statistically significant relationship between exposure and Plaintiffs' claimed injuries, or studies that show a statistically significant association only at dose levels that exceed Plaintiffs' exposure. *See Lipitor III*, 174 F. Supp. 3d. at 922–24.

There can be no dispute that "dose certainly matters" in this case. *Id.* at 919. Dr. Braun testified that there is a dose-response relationship between PFOS/PFOA and kidney cancer. JX 1

(Braun Dep.) at 106:11–14.[1] Thus, "Plaintiffs must have expert testimony" that PFOS or PFOA "causes or is capable of causing" kidney cancer at "particular dosages." *Lipitor III*, 174 F. Supp. 3d at 919. Because Dr. Braun falls well short of this mark, the Court should exclude his opinions.

1. Dr. Braun Cannot Point To Any Statistically Significant Association Between PFOS And Kidney Cancer.

Dr. Braun relied on just two observational studies to conclude [redacted]

The Shearer study looked at whether exposure to PFOS and PFOA is associated with an increased incidence of various forms of renal cell carcinoma, the most common form of kidney cancer. It compared the incidence of cancer in people who had elevated concentrations of PFOS or PFOA in their blood serum to those who had lower concentrations. *Id.*; JX 2 at 580. Dr. Braun claimed [redacted]

[1] "JX" refers to the joint exhibits filed concurrently in support of Defendants' Omnibus Motions to Exclude and Defendants' Motion for Summary Judgment, also filed concurrently.

[2] JX 2 (Joseph J. Shearer et al., *Serum Concentrations of Per- and Polyfluoroalkyl Substances and Risk of Renal Cell Carcinoma*, 113 J. Nat'l Cancer Inst. 580 (2021), Braun Dep. Ex. 21).

[3] JX 3 (Huiqi Li et al., *Cancer incidence in a Swedish cohort with high exposure to perfluoroalkyl substances in drinking water*, 204 Env't Rsch., 2022).

[4] Values at or below 1.0 are underlined throughout this brief to show confidence intervals that are not statistically significant.

[redacted]

"Attenuated" does not tell the story. The truth is that in every quartile (including the highest exposure group in the fourth quartile), the "'range of possible values' for the actual relevant risk ratio" included "values less than 1.0," which "do not show any increased risk, and indeed, show a decreased risk." *Lipitor III*, 174 F. Supp. 3d at 915. Specifically, the confidence intervals ranged from 0.59 to 2.57 for the second quartile of exposure, from 0.22 to 1.24 for the third quartile, and from 0.45 to 2.88 for the fourth quartile. JX 2 at 584, tbl. 2; *see also* JX 1 at 250:16–251:2. In other words, "the study does not demonstrate a 'statistically significant' increased risk of an adverse outcome." *Lipitor III*, 174 F. Supp. 3d at 915. Ultimately, Dr. Braun conceded that he should have considered the "fully adjusted odds ratio" for PFOS when he evaluated the Shearer study; he admitted that if he had used the correct data, "none of the fully adjusted results for PFOS in the Shearer study are statistically significant."[6] JX 1 at 249:1–23.

Dr. Braun's reliance on the Li study is just as deficient. Li studied cancer rates among

[5] This Court's explanation of the relationship between confidence intervals and risk ratios is instructive: "A confidence interval is essentially a 'margin of error' for the estimated relative risk ratio." *Lipitor III*, 174 F. Supp. 3d at 915. "So, for example, if a given study showed a relative risk of 1.40 (a 40 percent increased risk of adverse events), but the 95 percent confidence interval is .8 to 1.9, we would say that we are 95 percent confident that the true value, that is, the actual relative risk, is between .8 and 1.9." *Id.* "Because the confidence interval includes results which do not show any increased risk, and indeed, show a decreased risk, that is, it includes values less than 1.0, we would say the study does not demonstrate a 'statistically significant' increased risk of an adverse outcome." *Id.* And a confidence interval that includes 1.0 would also be not statistically significant, because a "relative risk of 1.0 indicates no difference between the two groups." *Id.*

[6] The first quartile in Shearer's study is the control group. *See* JX 2 at 580 (comparing risk ratios "among those in the highest quartile vs the lowest").

individuals in a Swedish municipality who were exposed to PFOS, PFOA, and PFHxS present in drinking water from AFFF. JX 3 at 1. Dr. Braun reported [redacted] And the Li results did not disaggregate PFOS from other PFAS compounds. *See* JX 3 at 1, 6, 7.

Dr. Braun was unable to point to any study that showed a relevant, statistically significant association between PFOS and kidney cancer.

2. Dr. Braun Found No Relevant Statistically Significant Associations Between PFOA And Kidney Cancer.

The same is true as to PFOA. In addition to the Shearer study (discussed above), which analyzed exposure to PFOA as well as to PFOS, Dr. Braun [redacted][7] Vieira examined the relationship between PFOA and cancer outcomes among residents living near a DuPont Teflon-manufacturing plant. JX 5 at 318. Neither the Shearer nor the Vieira study reported statistically significant results at the levels of PFOA to which

[7] [redacted]; JX 5 (Veronica M. Vieira et al., *Perfluorooctanoic Acid Exposure and Cancer Outcomes in a Contaminated Community: A Geographic Analysis*, 121 Env't Health Persp. 318 (2013)).

Plaintiffs' expert, Dr. MacIntosh, claimed Plaintiffs were exposed.

[redacted]

At six ppb or lower, the Vieira study did not show a positive association *at all*, let alone a statistically significant one. For the "low exposure" cohort that Plaintiffs would have been part of—i.e., people who had PFOA levels between 3.7 to 12.8 ppb—the Vieira study yielded a point estimate of 0.8 and a statistically insignificant confidence interval of 0.4 to 1.5, meaning there was no finding of increased risk of kidney cancer. JX 5 at 321, tbl. 2.

Vieira also did not find a statistically significant association in the next, "medium exposure" cohort, composed of people who had PFOA blood-serum levels between 12.9 and 30.7 ppb—i.e., a cohort that started at double the highest dose level estimated by Dr. MacIntosh and ran up to dose levels five times that high. *See id.* The confidence interval for the cohort exposed to 12.9 to 30.7 ppb was 0.7 to 2.0, meaning that there was not a statistically significant association between PFOA exposure and kidney cancer for that cohort. *Id.*

[8] Defendants separately challenge Dr. MacIntosh's blood-serum calculation methods in the concurrently filed Second Omnibus Motion to Exclude.

[9] For the Court's convenience, Appendix A [redacted]

It was not until exposure levels of [redacted], that Vieira found a statistically significant association. *See id.* Vieira found a confidence interval of 1.3 to 3.2 for the "high exposure" category of people with [redacted] are far lower than the "high" exposure category, which was the only exposure level at which Vieira found a statistically significant association.

And just as with PFOS, once Dr. Braun disaggregated the Shearer results to focus only on PFOA (in this case by removing PFOS and PFHxS), there was *no* statistically significant association, even for those with the highest levels of exposure to PFOA. The confidence interval ranged from 0.69 to 2.90 for the second quartile, from 0.52 to 2.42 for the third quartile, and from 0.86 to 5.61 for the fourth quartile. JX 2 at 584, tbl. 2. As Dr. Braun admitted, when looking at "the fully adjusted results for PFOA in Table 2 of the Shearer study," "none of the quartile results are statistically significant" because the "lower bounds" of the confidence intervals run from ".5 to approximately .9." JX 1 at 239:10–23.

Dr. Braun also conceded that the fully adjusted model was "preferable" in isolating the effect of PFOA. *Id.* at 235:7–238:14 (agreeing that PFOS would be a potential confounder if trying to isolate effect of PFOA and that it would be "more appropriate" to look at the fully adjusted model). And that is particularly true here, where Plaintiffs agree the Telomer Defendants could not have contributed to the presence of PFOS or PFHxS (though Plaintiffs also do not pursue claims based on PFHxS exposures). But even without disaggregating the results, the Shearer study still did not yield a statistically significant association relevant to the maximum potential exposure

level at issue in these cases (six ppb). [redacted]

Dr. Braun also attempted to pass off aggregated results as relevant by pointing to the Shearer study's "continuous variable" analysis, which evaluated *all* exposure levels combined together (rather than comparing different levels). JX 1 at 242:1–243:11. But given that (1) [redacted] and (2) according to Shearer there was no statistically significant association at that specific, known level, this generalized "continuous variable analysis" is irrelevant to the specific facts of these cases. *Lipitor III*, 174 F. Supp. 3d at 918 (evidence of association between Lipitor and diabetes at doses of 80 milligrams did not "support a causation opinion" at 10 milligrams where study showed the risk at lower doses "is meaningfully different from the risk at higher dose[s]"). Therefore, Dr. Braun cannot reasonably rely [redacted]

Dr. Braun [redacted] The Rhee study examined the relationship between PFOS and

PFOA and renal cell carcinoma in a multiethnic population.[10] Dr. Braun conceded at his deposition that the risk ratios from Rhee did not support an association between PFOA and kidney cancer. JX 1 at 258:24–259:22, 270:11–21. He acknowledged that the same was true of the Winquist study, which looked at PFOA exposure in participants enrolled in the American Cancer Society's Cancer Prevention Study.[11] Dr. Braun admitted that "none of the results" in the Winquist study "would meet conventional standards of statistical significance," including when one separately considered the results for females and males in the study. JX 1 at 286:12–17, 287:10–288:7.

As this Court held in *Lipitor III*, without a valid finding of association, Dr. Braun cannot even get to evaluating causation; his analysis fails at the very threshold. 174 F. Supp. 3d at 916, 926. That alone is grounds to exclude Dr. Braun's testimony. *See In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs. & Prods. Liab. Litig. ("Lipitor V")*, 892 F.3d 624, 640–42 (4th Cir. 2018).

B. Dr. Braun Failed To Conduct A Reliable Bradford Hill Causation Analysis.

The Court should also exclude Dr. Braun's opinion on general causation because [redacted] Under the Fourth Circuit's decision in *Lipitor V*, in order to "apply[] the

[10] JX 10 at 1 (Jongeun Rhee et al., *Serum concentrations of per- and polyfluoroalkyl substances and risk of renal cell carcinoma in the Multiethnic Cohort Study*, 180 Env't Int'l, 2023, Braun Dep. Ex. 22).

[11] JX 11 at 1 (Andrea Winquist et al., *Case-Cohort Study of the Association between PFAS and Selected Cancers among Participants in the American Cancer Society's Cancer Prevention Study II LifeLink Cohort*, 131 Env't Health Persp., 2023, Braun Dep. Ex. 23).

[12] "In assessing causation, epidemiologists 'first look for alternative explanations for the associations, such as bias or confounding factors,' and then apply the Bradford Hill factors to determine whether an association reflects a truly causal relationship." *Lipitor III*, 174 F. Supp. 3d

Bradford Hill criteria to a set of data," an expert must "find a *statistically significant* association at step one before moving on to apply the factors at step two." 892 F.3d at 642 (emphasis added); *see also Lipitor III*, 174 F. Supp. 3d at 924 ("it is well established that the Bradford Hill method used by epidemiologists *does* require that an association be established through studies with statistically significant results") (collecting cases). That is because under the Reference Manual on Scientific Evidence and case law, the Bradford Hill method is used to evaluate "whether an association shown by a study establishes causation." *Lipitor III*, 174 F. Supp. 3d at 925 (citing Reference Manual on Scientific Evidence 566 (3d ed. 2011)). Here, Dr. Braun did not find a statistically significant association between PFOS or PFOA at relevant exposures.

Dr. Braun's exclusive "reliance on non-statistically significant" findings was an epidemiologically unsound deviation from the norm. *Lipitor III*, 174 F. Supp. 3d at 926. Plaintiffs have failed to show that Dr. Braun's "reliance on non-statistically significant 'trends' is accepted in his field" or has "served as the basis for any epidemiologist's causation opinion in peer-reviewed literature." *Id.* To the contrary, Dr. Braun conceded that the most recent statement from the American Statistical Association that he was aware of reaffirmed the continuing importance of p-values and statistical significance. JX 1 at 102:12–103:15 (referencing JX 12 (Yoav Benjamini et al., *The ASA President's Task Force Statement on Statistical Significance and Replicability*, 15 Annals of Applied Stats. 1084 (2021), Braun Dep. Ex. 11)). In relying only on statistically insignificant results to draw a causal inference, Dr. Braun "unreliably applied the Bradford Hill methodology and thus his opinion" should be excluded. *Lipitor V*, 892 F.3d at 642.

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at 916. The Bradford Hill factors are: "(1) strength of the association, (2) replication of the findings, (3) specificity of the association, (4) temporal relationship, (5) dose-response relationship (aka biological gradient), (6) biological plausibility, (7) consistency with other knowledge (aka coherence), (8) consideration of alternative explanations, and (9) cessation of exposure." *Id.*

Dr. Braun's [redacted] But the Navigation Guide "is not designed to distinguish a causal connection from a mere association." *In re Acetaminophen*, 707 F. Supp. 3d 309, 341 (S.D.N.Y. 2023). [redacted] the Bradford Hill factors and the Navigation Guide stand "[i]n contrast," because Bradford Hill "address[es] questions of causation," whereas "the Navigation Guide is a tool used to summarize evidence," *In re Acetaminophen*, 707 F. Supp. 3d at 354. Experts cannot "offer reliable testimony of general causation without performing a reliable Bradford Hill analysis" independent of the Navigation Guide. *Id.* at 341. There can be no dispute that Dr. Braun did not conduct an independent Bradford Hill analysis.

Indeed, *In re Acetaminophen* is on all fours. There, the court found that the expert "cherry-picked and misrepresented study results" and that his "grading of the studies, examined closely, shows [] evidence of 'result-driven analysis.'" *Id.* at 339, 355 (quoting *Lipitor V*, 892 F.3d at 634). In particular, the court observed that the expert opportunistically and inconsistently rated studies based on whether they supported or undermined his favored conclusion. *Id.* at 355. That was "a paradigmatic example of interpreting results differently based on the outcome of the study" and was "illustrative" of the expert's "approach to the Navigation Guide, in which he use[d] areas when an expert's subjective opinion comes into play to selectively downgrade studies not supporting his analysis and vice versa." *Id.*

Dr. Braun's application of the Navigation Guide methodology was similarly flawed and unreliable. The Court should exclude this analysis for at least three reasons.

1. Cherry-Picked Sources. Dr. Braun "fail[ed] to meaningfully account for . . . literature

at odds with [his] testimony." *Lipitor III*, 174 F. Supp. 3d at 920; *id.* at 929–33, 935 (Experts must "review the totality of the literature"—as if "conducting a literature review for publication"—so "cherry-picking studies that support [the expert's] conclusion" is unreliable and "fails to satisfy the scientific method and Daubert."). He omitted multiple published studies that met his inclusion criteria but that undermined his conclusion that there is an association between PFOS and PFOA and kidney cancer. For example, Dr. Braun omitted a 2024 study by Alexander that "did not report an association with kidney cancer and total PFOS equivalents."[13] Dr. Braun failed to give any reason for neglecting to include it. JX 1 at 195:4–199:4.

And Dr. Braun failed to grapple with the EPA's warning that there is "limited mechanistic information that could contribute to the determination of a causal relationship" between PFOS or PFOA and kidney cancer.[14] He also failed to address the EPA's conclusion that "convincing epidemiological evidence supporting a causal association between human exposure to PFOA and cancer are lacking." *Id.* at 15; *id.* at 24 (same for PFOS). Dr. Braun similarly did not mention, let alone address, the Agency for Toxic Substances and Disease Registry's (ATSDR) repeated conclusion in 2019[15], 2021[16], and most recently in 2024 that "[c]ausal relationships have not been established for these health effects [including kidney cancer]."[17]

2. Results-Driven Quality Ratings. In subjectively rating the quality of the studies he

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[13] JX 13 at 10 (Bruce H. Alexander et al., *Mortality and cancer incidence in perfluorooctanesulfonyl fluoride production workers*, Am. J. Indus. Med., 2024, Braun Dep. Ex. 19).

[14] JX 14 at 16, 24–25 (EPA, *Maximum Contaminant Level Goals for Perfluorooctanoic Acid (PFOA) and Perfluorooctane Sulfonic Acid (PFOS) in Drinking Water*, Apr. 2024).

[15] JX 15 at 8 (Excerpt of ATSDR, *PFAS: an overview of the science and guidance for clinicians on per- and polyfluoroalkyl substances (PFAS)*, Dec. 6, 2019).

[16] JX 16 at 751 (Excerpt of ATSDR, *Toxicological Profile for Perfluoroalkyls*, May 2021).

[17] JX 17 at 4 (ATSDR, *Health Effects: PFAS Information for Clinicians – 2024*, Nov. 12, 2024).

did consider, Dr. Braun granted a higher rating to studies that supported his desired conclusion—an association between kidney cancer and PFAS—and downgraded to a lower rating studies that contradicted his desired conclusion.[18] In fact, at his deposition, Dr. Braun [redacted] But at deposition, Dr. Braun admitted that he should have rated the Law study at least the same as the Mastrantonio study. JX 1 at 150:4–14.

3. Selective Use of Study Data. Dr. Braun also cherry-picked favorable portions of studies while discounting aspects of the same studies that undermined his conclusions. For instance, to support the opinion that PFOA is associated with kidney cancer, Dr. Braun relied on the above-discussed Rhee study. [redacted]

[18] The subjectivity and opportunism inherent in Dr. Braun's Navigation Guide analysis are exacerbated by his admitted failure to conduct that analysis alongside additional researchers, which Dr. Braun conceded to be a feature of a reliable Navigation Guide analysis, JX 1 at 131:7–11, and one that was present in *every* Navigation Guide analysis on which Dr. Braun relied, *id*. at 132:1–6. *See In re Acetaminophen*, 707 F. Supp. 3d at 354 (the Navigation Guide "is intended to be used by teams," so as to "minimize bias in the evaluation of the evidence").

[19] JX 18 (Marina Mastrantonio et al., *Drinking water contamination from perfluoroalkyl substances (PFAS): an ecological mortality study in the Veneto Region, Italy*, Eur. J. Pub. Health, 2023, Braun Dep. Ex. 13).

[20] JX 19 (HD Law et al., *Relative rates of cancers and deaths in Australian communities with PFAS environmental contamination associated with firefighting foams: A cohort study using linked data*, 82 Cancer Epidemiology, 2023, Braun Dep. Ex. 14).

[redacted]

[redacted]

These same flaws doom Dr. Braun's use of the Navigation Guide even to find an association between PFOS and PFOA and kidney cancer, let alone as a substitute for the required Bradford Hill analysis. Considering "the entire process that produced" Dr. Braun's opinions, it is evident that his work does not "satisf[y] *Daubert*'s fundamental command: that expert testimony be reliable and relevant." *Lipitor V*, 892 F.3d at 638. His flawed use of the Navigation Guide, substituted for a Bradford Hill analysis, has all the hallmarks of expert testimony that should be excluded. His opinions were "results driven." *Id.* at 634. He did not "examin[e] 'the literature as a whole'" and instead "ignore[d] . . . information and research" that did not support his opinions. *Hoefling v. U.S. Smokeless Tobacco Co.*, 576 F. Supp. 3d 262, 273 (E.D. Pa. 2021). And he "'cherry-pick[ed]' relevant data" such that his approach "does not reflect scientific knowledge, is not derived by the scientific method, and is not 'good science.'" *Lipitor V*, 892 F.3d at 634.

C. Dr. Braun's Opinion That "Any Amount" Of Exposure To PFOS And PFOA Increases The Risk Of Kidney Cancer Is Unreliable.

Faced with the reality that the epidemiological literature does not support a statistically significant association between PFOS (at all) or PFOA (at relevant levels of exposure), Dr. Braun resorted to the unsound and oft-rejected conclusion that [redacted] This "any

[22] *See* JX 11 at 5 (RR: 1.33; 95% CI: 0.97, 1.83); *see also* JX 1 at 287:6–16 (confirming results and agreeing they are not statistically significant).

exposure" opinion is demonstrably unreliable.[23]

Courts across the country—including this Court and the Fourth Circuit—have time and again rejected opinions that any amount of a substance is capable of causing harmful effects. *See, e.g.*, *Lipitor V*, 892 F.3d at 639; *In re: Lipitor (Atorvastatin Calcium) Mktg. ("Lipitor I")*, 2015 WL 6941132, at *2 (D.S.C. Oct. 22, 2015); *In re Deepwater Horizon BELO Cases*, 119 F.4th 937, 941 (11th Cir. 2024); *Betz v. Pneumo Abex, LLC*, 44 A.3d 27, 49 n.25, 55–56 (Pa. 2012) (excluding expert's opinion that "any exposure" to asbestos was sufficient to cause mesothelioma on ground that the "no safe level or linear 'no threshold' model . . . 'flies in the face of the toxicological law of dose-response,'" and is "in irreconcilable conflict with itself" because if "each and every exposure" is a "substantial contributing cause," then no exposure can be insubstantial).

Dr. Braun did not cite a single study with statistically significant results to support his conclusion that [redacted] *see also* JX 1 at 343:6–8. Dr. Braun ignored these realities by inverting the burden of proof—concluding that there is an increased risk at any dose because, he claims, there is no affirmative evidence that there is not. Of course, this is impossible to square with Dr. Braun's analysis [redacted] But Dr. Braun blew past that, arguing that, because increased risk was found at higher exposure levels and because in his view there is a dose-response relationship, there must be an increased risk at lower exposure levels, all the way to zero. JX 1 at 210:16–217:7, 218:21–224:15. That *ipse dixit* and illogical reasoning

[23] Defendants' concurrently filed motion for summary judgment discusses rejected theory of "any exposure" in depth. *See* Motion for Summary Judgment, Section I.A.

violates the toxicological axiom of dose-response, *Betz*, 44 A.3d at 49 n.25, and is directly at odds with this Court's reasoning that a "risk estimate from a study that involved a greater exposure is not applicable to an individual exposed to a lower dose," *Lipitor V*, 174 F. Supp. 3d at 921.

Because there is no scientific support for Dr. Braun's opinion that any exposure to PFOS or PFOA is capable of causing Plaintiffs' cancer, the Court should exclude that opinion.

II. Dr. Bahnson's Specific Causation Opinions Should Be Excluded.

The Court should also exclude the testimony of Plaintiffs' sole specific causation expert, Dr. Bahnson, under Rule 702. His opinions are inadmissible for at least three reasons:

First, Dr. Bahnson's opinions [redacted] He did not consider relevant data and failed to apply a reliable scientific methodology. And he admittedly failed to consider the [redacted]

Second, Dr. Bahnson's opinion [redacted]

Third, Dr. Bahnson's opinions that [redacted]

A. The Court Should Exclude Dr. Bahnson's Specific-Causation Testimony Because His Differential Etiology Is Unreliable.

To establish specific causation, Plaintiffs must offer admissible expert testimony "utilizing a valid methodology, applying it reasonably and relying on sufficient data to support her opinions"

that concludes "to a reasonable degree of medical certainty that the particular plaintiff is in the minority of those that developed the disease due to exposure to a particular drug or substance rather than in the majority that would have developed the disease regardless." *In re Lipitor (Atorvastatin Calcium) Mktg., Sales Pracs. & Prods. Liab. Litig. ("Lipitor II")*, 150 F. Supp. 3d 644, 650 (D.S.C. 2015). Here, Dr. Bahnson's methodologies were not "valid" or applied "reasonably," and he did not "rely[] on sufficient data." *Id.* Thus, his specific causation opinions are inadmissible.[24]

In arriving at the conclusion that PFOS or PFOA was the specific cause of Plaintiffs' kidney cancer, Dr. Bahnson purported to apply a differential etiology method, which required him "to determine whether a particular source is the likely cause of an individual's disease" by "eliminating the likely causes until the most probable one is isolated." *Lipitor V*, 892 F.3d at 643.[25] A reliable differential etiology "must at least consider other factors that could have been the sole cause of the plaintiff's injury" and "offer an explanation as to why these other recognized causes, alone, are not responsible for the disease in a particular plaintiff." *Id.* at 643–44. The expert must take "serious account" of those "other potential causes." *Id.* at 644 (quoting *Westberry v. Gislaved Gummi AB*, 178 F.3d 257, 265 (4th Cir. 1999)). Where a plaintiff's "symptoms could have numerous causes," the expert cannot "simply pick[ ] the cause that is most advantageous to [plaintiff's] claim." *Viterbo v. Dow Chem. Co.*, 826 F.2d 420, 424 (5th Cir. 1987).

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[24] Plaintiffs make no pretense that they can establish specific causation by having an expert witness "testify to specific causation based on epidemiologic studies that find a relative risk of injury of 2.0 or higher and what is referred to as 'the logic of the effect of doubling of the risk.'" *Id.* at 649 (citing Reference Manual on Scientific Evidence 612 (3d ed. 2011)). Plaintiffs cannot identify any study showing a two-fold increased risk of kidney cancer from exposure to PFOS or PFOA at relevant concentrations. [redacted]

[25] The term "differential etiology" is "used to describe the process by which the cause of an injury is determined." *Lipitor II*, 150 F. Supp. 3d at 660 n.17. However, the trend in federal courts is to use the term "differential diagnosis." *Id.*

[redacted]

Rather than "offer an explanation as to why [the other risk factors] . . . are not responsible for" each of the Plaintiffs' kidney cancer, *Lipitor V*, 892 F.3d at 643–44, as discussed below, Dr. Bahnson automatically ruled out recognized risk factors in all cases. Then, as to each Plaintiff, he [redacted] *See Lipitor II*, 150 F. Supp. 3d at 661. Dr. Bahnson did not even [redacted] as he was required to do under this Court's reasoning. *See id.* at 656–57.

That is "closer to an *ipse dixit* than a reasoned scientific analysis." *Lipitor V*, 892 F.3d at 645; *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997) (appropriate to exclude "opinion evidence that [was] connected to existing data only by the *ipse dixit* of the expert"). Because there is too great an "analytical gap" between the data and Dr. Bahnson's conclusions ruling out recognized causes of kidney cancer, his opinions should be excluded. *See Joiner*, 522 U.S. at 146.

1. Dr. Bahnson Provided No Reliable Basis for Concluding that Excess Body Weight And Hypertension Are Not Causes Of Plaintiffs' Kidney Cancer.

Dr. Bahnson's opinions [redacted]

[redacted]

There is no dispute: [redacted] JX 27 at 2 (American Cancer Society, *Kidney Cancer Causes, Risk Factors, and Prevention* (last revised May 1, 2024)); [redacted]

Yet, Dr. Bahnson admitted [redacted]

[26] *See also* JX 28 (Furan Wang & Yinghua Xu, *Body mass index and risk of renal cell cancer: a dose-response meta-analysis of published cohort studies*, 135 Int'l J. Cancer 1673 (2014)); JX 29 (Khemayanto Hidayat et al., *Blood pressure and kidney cancer risk: meta-analysis of prospective studies*, 35 J. Hypertension 1333 (2017)).

[redacted]

[redacted]

This flips the differential etiology methodology on its head; [redacted] *Cooper v. Smith & Nephew, Inc.*, 259 F.3d 194, 202–03 (4th Cir. 2001) (affirming exclusion of expert testimony where expert "did not identify how he ruled out smoking and other potential causes" of the injury and simply "read two articles on smoking and rejected them as unpersuasive").

Dr. Bahnson could not [redacted] "The first factor identified by *Daubert* as a 'key question' in determining whether a technique can be considered reliable scientific knowledge is whether it has been tested and independently validated or replicated." *Ruffin v. Shaw Indus., Inc.*, 149 F.3d 294, 297 (4th Cir. 1998). Dr. Bahnson [redacted] And without "adequate validation," the opinion is not

[redacted]

trustworthy. *Westberry*, 178 F.3d at 260.

Dr. Bahnson was unable [redacted]

To perform a valid, reliable differential etiology, Dr. Bahnson had to actually take known risk factors into account, and not just summarily deny that they were risk factors. This Court made that clear in *Lipitor II*, where the "Plaintiff had a *number of statistically significant risk factors* for diabetes beyond ingestion of Lipitor, including her BMI, recent weight gain, total adult weight gain, age, family history, hypertension, and possibly metabolic syndrome." 150 F. Supp. 3d at 661 (emphasis added). This Court excluded one of the plaintiff's specific-causation experts (Dr. Murphy) because although she labeled her methodology a "differential diagnosis," she "acknowledged that she had no method to identify the minority of Lipitor users whose development of diabetes while on the medication was caused by Lipitor, *as opposed to other risk factors*." *Id.* (emphasis added); *see also In re Roundup Prods. Liab. Litig.*, 2025 WL 869146, at *1–2 (N.D. Cal. Mar. 14, 2025) (excluding expert's opinion that did not consider the plaintiffs' increased weight, despite it carrying a "statistically significant" increased risk for non-Hodgkin

[29] JX 30 (AH Mokdad, et al., *The Spread of the Obesity Epidemic in the United States, 1991-1998*, 282 JAMA, 1999).

lymphoma, along with other exposures). Here too, Dr. Bahnson's failure to take known risk factors into account dooms his opinions.

2. Dr. Bahnson Provided No Basis For Ruling Out Hereditary Genetic Mutations As The Cause Of Plaintiffs' Kidney Cancer.

Similarly, Dr. Bahnson did not [redacted] *Lipitor V*, 892 F.3d at 644.

Kevin Voelker. Mr. Voelker [redacted]

[redacted]

Dr. Bahnson's opinion is contrary to cancer research. Indeed, multiple studies have found a statistically significant association between the c.1100del variant of the CHEK2 mutation and kidney cancer, with odds ratios ranging from 3.61[31] to 9.8[32]—far higher than any elevated risks reported for PFOS or PFOA at Plaintiffs' exposure levels (all below 2.0 with statistically insignificant confidence intervals, *see supra*, at 3–9, 17 n.24). There is thus "too great an analytical gap" between the admitted relationship between kidney cancer and the CHEK2 variant, and Dr. Bahnson's opinion ruling it out as an alternative cause. *See Casey v. Geek Squad Subsidiary Best Buy Stores, L.P.*, 823 F. Supp. 2d 334, 347 (D. Md. 2011) (excluding testimony where expert identified no accepted "facts or theory . . . regarding the causes of electrical shock by a personal computer to support his conclusion"); *Cooper*, 259 F.3d at 202–03 (opinion that smoking was not a cause of injury was contrary to "the medical literature").

Clinton Speers. Dr. Bahnson likewise [redacted]

[31] JX 31 at 1 (Charlotte Naslund-Koch et al., *Increased Risk for Other Cancers in Addition to Breast Cancer for CHEK2*1100delC Heterozygotes Estimated From the Copenhagen General Population Study*, 34 J. Clinical Oncology 1208 (2016)) (95% CI: 1.33, 9.79).

[32] JX 32 at 59, tbl. 2 (Maren Weischer et al., *Increased risk of breast cancer associated with CHEK2*1100delC*, 25 J. Clinical Oncology 57 (2007)) (95% CI: 2.3, 41.2).

[redacted]

For both [redacted] Dr. Bahnson failed to seriously consider them or "to offer an explanation for why the proffered alternative cause was not the sole cause," and thus his opinions must be excluded. *Cooper*, 259 F.3d at 202.

3. Dr. Bahnson's Opinion Ruling Out TCE As The Cause Of Plaintiffs' Kidney Cancer Is Unreliable.

Additionally, Dr. Bahnson's differential etiology is unreliable because [redacted]

The military bases at issue and surrounding areas have long experienced contamination from Volatile Organic Compounds ("VOCs"), including TCE. [redacted]

The IARC has concluded that "[t]richloroethylene causes cancer of the kidney" in

[33] JX 33 (Claire Forde et al., Hereditary Leiomyomatosis and Renal Cell Cancer: Clinical, Molecular, and Screening Features in a Cohort of 185 Affected Individuals, 3 Eur. Urology Oncology 764 (2020)); *see also* [redacted]

humans.[34] JX 37 at 189. Likewise, the ATSDR has concluded that "[t]here is strong evidence that trichloroethylene can cause kidney cancer in people."[35]

Dr. Bahnson agreed, [redacted] Both reasons are results-driven and unreliable.

Dr. Bahnson's first rationale is a paradigmatic example of a double standard. Dr. Bahnson [redacted] In light of this inconsistent approach, Dr. Bahnson offered [redacted] *In re Roundup Prods. Liab. Litig.*, 2025 WL 869146, at *1.

As to his other rationale, Dr. Bahnson cited [redacted]

[34] JX 37 (IARC, *TRICHLOROETHYLENE, TETRACHLOROETHYLENE, AND SOME OTHER CHLORINATED AGENTS*, 106 IARC Monographs, 2014).

[35] JX 62 at 4 (ATSDR, *Toxicological Profile for Trichloroethylene*, June 2019, excerpted).

First, there was no "regulatory" standard for TCE in drinking water until 1989, when EPA imposed a maximum contaminant level. *See* JX 39 (EPA Consumer Fact Sheet).

Second, Mr. Brown did not opine

[36]

Thus, Dr. Bahnson's decision

Dr. Bahnson also

[36] For example, a 1980 EPA report indicated that six of fifteen wells in Warminster Township were contaminated with PCE and TCE, with levels as high as 260 ppb (more than fifty times the later-enacted MCL). JX 40 at 26–27 (Excerpt of EPA, *Final Environmental Impact Statement: Horsham-Warminster-Warrington, Pennsylvania Wastewater Treatment Facilities*, May 7, 1980, Brown Dep. Ex. 12).

[redacted] *Lipitor V*, 892 F.3d at 644.

4. Dr. Bahnson Provided No Basis For Ignoring Natural DNA Replication Errors As The Cause Of Plaintiffs' Kidney Cancer.

Dr. Bahnson conceded [redacted] *Lipitor II*, 150 F. Supp. 3d at 661. [redacted] *See Lipitor V*, 892 F.3d at 643–44 (affirming exclusion of specific-causation testimony for failure to consider recognized causes); *see also Cooper*, 259 F.3d at 202 ("if an expert utterly fails to consider alternative causes," then "a district court is justified in excluding the expert's testimony").

For these reasons, Dr. Bahnson's differential diagnosis as to each Plaintiff is unreliable and should be excluded under Rule 702.

[redacted]

B. The Court Should Exclude Dr. Bahnson's Opinion Because It Depends On Dr. Braun's Inadmissible General Causation Opinion.

Dr. Bahnson does [redacted] To the extent Dr. Bahnson purported to offer opinions "ruling in" PFOS and PFOA as capable of causing kidney cancer, the Court should exclude those opinions as well. Dr. Bahnson [redacted] Dr. Bahnson's "unblinking reliance" on Dr. Braun's testimony without doing anything "to corroborate or validate" it renders Dr. Bahnson's opinions independently inadmissible. *Funderburk v. S.C. Elec. & Gas Co.*, 395 F. Supp. 3d 695, 721–22 (D.S.C. 2019). And Dr. Bahnson's [redacted][38]

C. Dr. Bahnson's Opinions About Increased Risks Of Additional Cancers And Other Health Conditions Should Be Excluded Because They Are Unreliable.

Dr. Bahnson opined [redacted]

[38] [redacted] *with* JX 41 at 16–17 (Sheila Zahm, et al., *Carcinogenicity of perfluorooctanoic acid and perfluorooctanesulfonic acid*, 25 Lancet Oncology 16 (2004), Bahnson Dep. Ex. 10) (the two-page summary stating that the IARC *could not conclude* that PFOS and PFOA cause kidney cancer), *and* JX 42 at 717–18 (IARC, *PERFLUOROOCTANOIC ACID (PFOA) AND PERFLUOROOCTANESULFONIC ACID (PFOS)*, 135 IARC Monographs, 2025, Bahnson Dep. Ex. 11) (full monograph with same conclusion).

[redacted]

[redacted] The Court should exclude his opinions about Plaintiffs' supposed future risks on that basis.

The Court should also exclude Dr. Bahnson's prognostic opinions because they are not based in scientific evidence [redacted] These opinions should be excluded.

III. Because Dr. Sherman's "Plausible" Biological Mechanism Opinion Is Scientifically Unsupported And Contradicted By Data He Ignores, It Is Unreliable And Inadmissible.

Dr. Sherman [redacted] This concentration

disparity makes his opinion unreliable and requires exclusion under Rule 702.

Additionally, Dr. Sherman's opinion [redacted] And he did not even consider that multiple FDA-approved medications that bind to and activate PPAR have been used for decades to treat chronic medical conditions, supported by epidemiological studies finding no increased risk of cancer.

A. Dr. Sherman's Opinion That PFOS And PFOA May Activate PPAR At Human-Relevant Concentrations Is Unsupported.

Dr. Sherman [redacted]

Specifically, [redacted][39] Ignoring a disparity of this

[39] [redacted]

magnitude is the very definition of an unreliable opinion. *See Baker v. Chevron USA, Inc.*, 680 F. Supp. 2d 865, 881–82 (S.D. Ohio 2010) (excluding testimony of expert because a plaintiff's "cumulative benzene exposure was anywhere from 14 to 42 times less than the lowest cumulative benzene exposure associated with leukemia").

Dr. Sherman admitted that he is [redacted] This concession alone is fatal to Dr. Sherman's opinion.

At his deposition, Dr. Sherman [redacted] In short, this was nothing more than *ipse dixit* backpedaling.

[redacted]

B. Even Setting Aside This 1,000x Concentration Disparity, Dr. Sherman Cannot Show That PPAR Activation Can Cause Kidney Cancer.

The second half of Dr. Sherman's [redacted]

On the contrary, the extensive clinical and epidemiological data show that use of FDA-approved PPAR agonist medications—which bind to and activate PPAR, exactly what Dr. Sherman claims PFOS and PFOA do—have not been shown to increase the rate of cancer in humans. As Dr. Sherman acknowledged [redacted]

Ignoring relevant data is not consistent with the scientific method or Dr. Sherman's own claim to have considered all the evidence pertinent to his hypothesis. *In re Rezulin Prods. Liab. Litig.*, 369 F. Supp. 2d 398, 425 (S.D.N.Y. 2005) ("[I]f the relevant scientific literature contains evidence tending to refute the expert's theory and the expert does not acknowledge or account for that evidence, the expert's opinion is unreliable."). Dr. Sherman has no support for his opinion that PPAR activation can cause kidney cancer. His unsupported opinions should be excluded.

CONCLUSION

The Court should exclude the opinions of Dr. Braun, Dr. Bahnson, and Dr. Sherman.

Dated: June 17, 2025

Respectfully submitted,

/s/ Brian Duffy

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Defense Co-Leads

Appendix A: Relevant Quartiles for Plaintiffs in Studies Dr. Braun Identified [redacted]

[redacted]

*A value of 1.00 indicates that no increased risk has been found. No confidence interval is listed because plaintiff's estimated dose falls within the lowest quartile against which higher-exposed associations were measured.

[1] All studies measured quartiles in ppb (or µg/L).

[2] Vieira at 321, tbl. 2.

[3] Rhee at 5, tbl. 2 (fully adjusted results).

[4] Shearer at 584, tbl. 2 (fully adjusted results).

[5] Winquist at 7, tbl. 3 (quartiles); *id.* at Supplemental Table S9 (results).

CERTIFICATE OF SERVICE

On June 17, 2025, I electronically submitted the foregoing document with the Clerk of Court, using the electronic case filing system of the Court. I hereby certify that I have served all parties electronically or by another manner authorized by Federal Rule of Civil Procedure 5(b)(2).

/s/ Brian Duffy
Brian Duffy